

etiology is only partially understood. Preliminary results on the use of inositol as a lipotropic agent have not shown a beneficial effect (26). Niacin feeding after calving showed some reduction in weight loss (14), but niacin has not been evaluated specifically as a preventive for fat cow syndrome.

GRASS TETANY

by H. F. Mayland

INTRODUCTION

Grass tetany (hypomagnesemia) is a major health problem of cattle and sheep in temperate climates. It is caused by a deficiency of utilizable magnesium (Mg). This nutritional disorder includes a number of clinical diseases known as grass tetany, grass staggers, crested wheatgrass poisoning, wheat pasture poisoning, winter tetany, transport tetany, pasture flush staggers, and in calves, a disorder called milk tetany. The problem occurs in the United Kingdom, western Europe, South Africa, Argentina, New Zealand and the USA.

Animal losses vary from year to year. In the United Kingdom it is estimated that clinical cases occur in about 1% of the animals and that one-third of these die (50). Losses may be similar in other countries where dairy cows are maintained on pasture with little or no supplemental feeding. Beef-cattle losses probably occur at a lower rate, because milk production and subsequent Mg losses are less than for dairy cows.

In the USA grass tetany is more common in beef cows and less common in dairy cows because many of the dairy animals are dry

lotted or stall fed with high quality forages and concentrates. Significant losses to grass tetany occur in sheep and may be similar to, or less than, losses in beef cattle under comparable conditions. Few attempts have been made to evaluate the effects of hypomagnesemia on production. However, increased milk production has been measured after oral Mg supplementation in New Zealand dairy herds where average blood Mg levels were previously 10 to 14 mg/l (47, 52).

Grass tetany has been investigated extensively, but the complex etiology is not well understood. Excellent reviews are available on the effects of soil, climate, forage, and animal interactions leading to hypomagnesemia. Of special interest are the two volumes dealing with broad aspects of grass tetany edited by Rendig and Grunes (33) and Fontenot et al (7).

ROLE OF Mg IN ANIMALS

Magnesium is essential for both animals and plants. It is the metal cofactor of many enzymes involved in the metabolism of carbohydrates, lipids and proteins. In animals it also exerts a strong influence on neuromuscular function. While Mg is essential to animals, it appears that there is no regulatory mechanism. Nevertheless, problems of Mg metabolism are rare in simple-stomached animals. However, Mg deficiency is common to ruminants.

The bodies of domestic ruminants contain about 0.05% Mg by weight (Fig. 24-5). Of that amount, about 65-70% is in bone, 15% in muscle, 15% in other soft tissues and 1% in extracellular fluids (8, 13, 23).

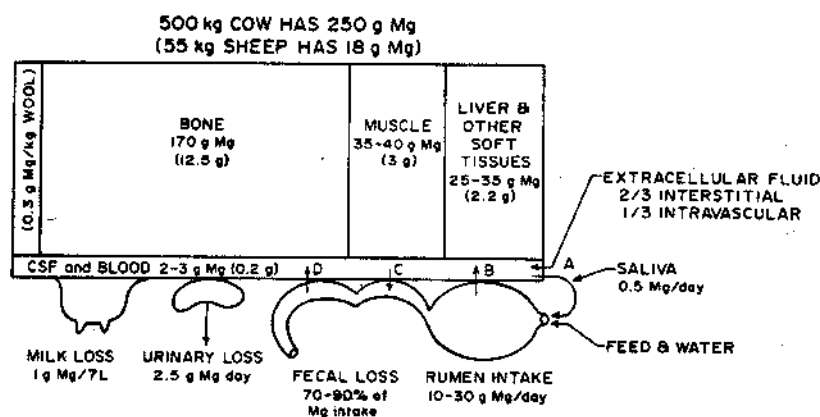


Figure 24-5. Model illustrating the distribution and movement of Mg in the cow (values for sheep are in parenthesis) including: (A) about 0.5 g returning in saliva, (B) primary absorption occurring from the rumen, (C) a small amount of resorption occurring in the small intestine, and (D) a small amount of absorption occurring from the large intestine.

Table 24-4. The amounts of Mg associated with the urinary and fecal endogenous loss, growth, pregnancy and lactation in sheep and cattle.^a

	Sheep	Cattle
Endogenous loss (Inevitable loss)	3 mg Mg/kg liveweight	3 mg Mg/kg liveweight
Growth	0.41 g Mg/kg gain	0.45 g Mg/kg gain
Lactation	0.17 g Mg/kg milk	0.12 g Mg/kg milk
Pregnancy (Daily increment - early in conceptus)	0.01 g Mg/d	0.12 g Mg/d
-mid	0.03 g Mg/d	0.21 g Mg/d
-late	0.05 g Mg/d	0.33 g Mg/d

^aFrom Grace (8)

Grace (8) summarized the absolute amounts of Mg needed for various production functions in sheep and cattle (Table 24-4). The data were developed from balance studies, isotope experiments and slaughter data.

The amount of Mg required by ruminants also depends upon animal age, size, stage of pregnancy, level of lactation and weather conditions (46). For example, mature pregnant beef cows fed a dry semipurified diet required 8.5, 7.0 and 9.0 g Mg/d to maintain the blood serum Mg level at 20 mg/l at 155, 200 and 255 d gestation, respectively (30). These cows then required 21, 22 and 18 g Mg/d during early, mid and late lactation to maintain the blood-serum Mg level at 20 mg/l, respectively (31). These experimentally derived values are greater than those that would be calculated from the information in Table 24-4, thus illustrating the low availability of Mg from these particular diets. Therefore, to calculate the Mg requirement on a dietary basis requires information on the availability of ingested Mg and its subsequent utilization by the animal.

FACTORS AFFECTING UTILIZATION OF Mg

Grass tetany occurs when animals do not ingest a sufficient amount of available Mg. This may occur when animals are on nutritionally poor diets, especially those low in Mg and Ca. However, grass tetany generally occurs when the dietary intake of total Mg is not particularly low, but factors exist (Fig. 24-6) which increase the animal's requirement for Mg or reduce the availability of dietary Mg to the

animal. Most commonly, grass tetany occurs during early lactation in older animals that have been turned out to spring pasture.

Forage quality may vary from pasture to pasture. Jolley and Leaver (16) reported that pasture on which grass tetany occurred (prone) contained lower concentrations of Mg, Ca and Na and higher concentrations of K than pastures on which tetany had not occurred (free).

Pasture	Mg	Ca	K	Na
 mg/g			
Prone	1.8	4.9	25	1.2
Free	2.2	5.3	22	2.8

They also noted that the grass tetany-prone pastures had a lower proportion of legume in the grass-legume mix.

Grass tetany is often associated with intensively managed cool-season grasses. Grasses have lower concentrations of Mg and Ca and higher concentrations of K than do legumes or forbs. This is illustrated by data obtained by the author from a large number of native and introduced plants sampled in Idaho, Nevada and Utah during midspring.

Plant Type	Mg	Ca	K
 mg/g		
Grasses	2	4	25
Legumes	3	14	20
Forbs	7	15	15

Grass tetany may occur on grass-legume mixed pastures, but does so in early spring when average air temperatures are <14°C (57°F). At that time the forage mixture is still mostly grass, because legume growth is limited by cool air temperatures. Cool soil temperatures can also result in forage Mg levels that are lower in spring than later when soil temperatures are warmer (Fig. 24-7).

Potassium

The Mg concentrations in forage and subsequently in the blood serum of cattle are influenced strongly by high amounts of fertilizer K and, to some extent, fertilizer N. This is illustrated by the following blood serum Mg

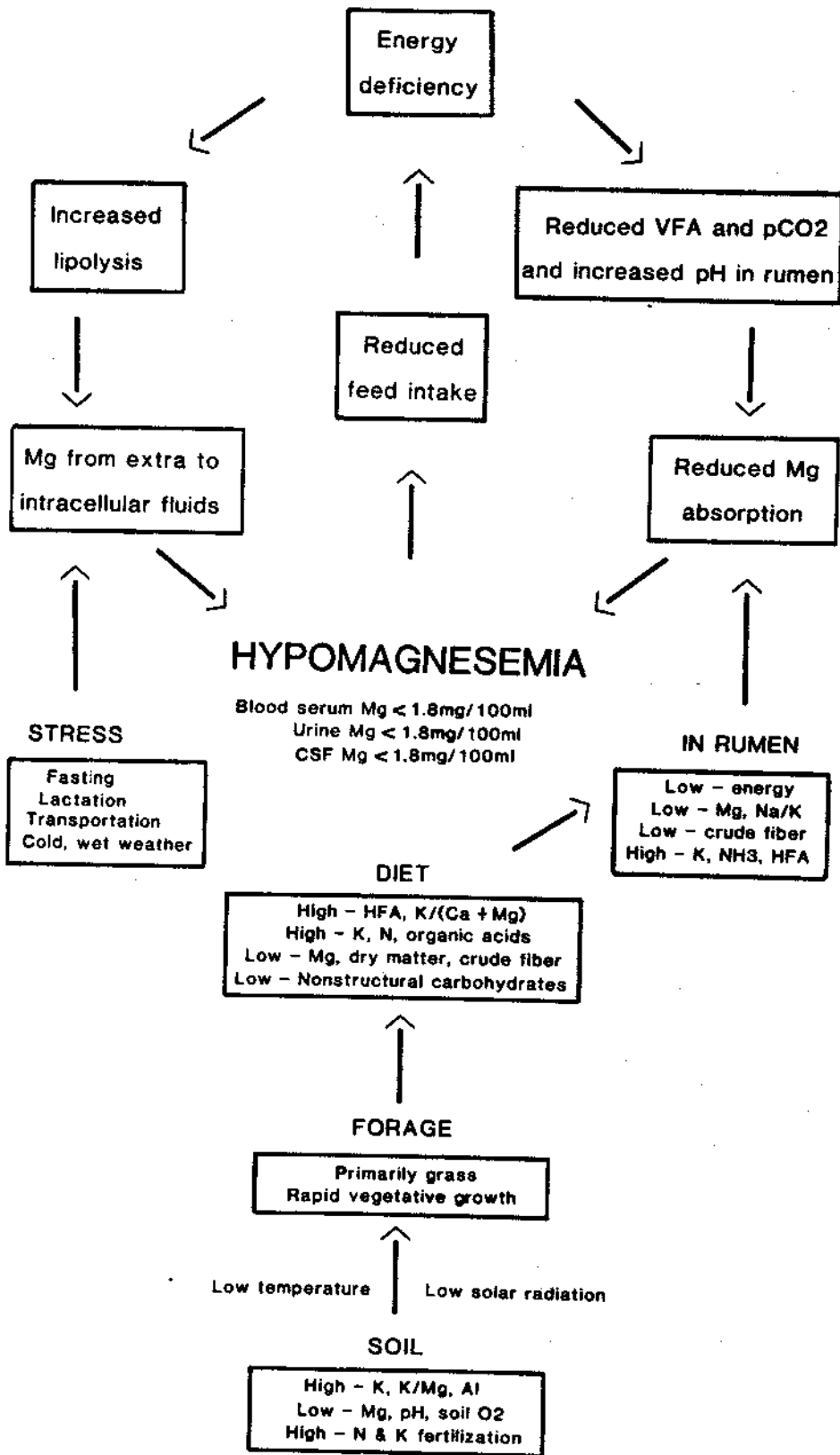


Figure 24-6. Schematic of the etiology of hypomagnesemia.

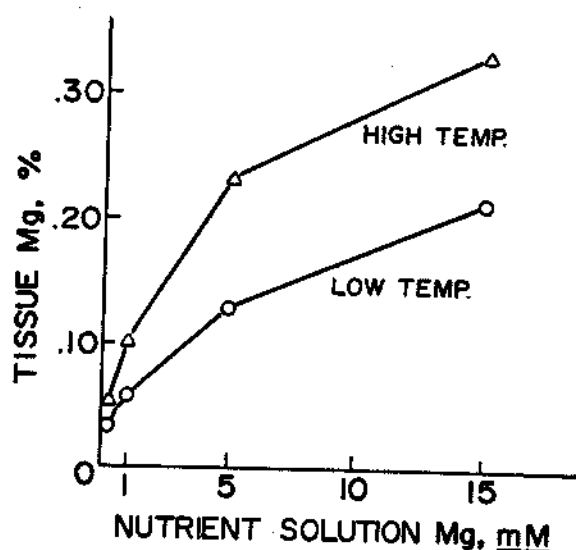


Figure 24-7. Tissue Mg levels in perennial ryegrass grown at 20°C day, 14°C night (low temp.) or 26°C day, 23°C night (high temp.) in sand culture containing given levels of Mg. Adapted from Grunes et al (11).

values in four dairy cows that grazed a series of differentially fertilized pastures (51). Similar findings have been reported for sheep.

Low N — low K	24.3 mg Mg/l
High N — low K	21.5 mg Mg/l
Low N — high K	17.4 mg Mg/l
High N — high K	14.1 mg Mg/l

Mg absorption by plants is reduced by high soil-K levels (24). Mg absorption by ruminants is also reduced by a high intake of K. Newton et al (29) fed wether lambs either a low-K (0.6%) or a high-K (4.9%) diet. Apparent absorption of Mg was depressed by the high-K relative to the low-K diet during eight consecutive 3-d trials (Fig. 24-8).

Sodium:Potassium

The Na concentration of immature grass is often insufficient to meet the requirements of the animal. This reduction (from dry winter diets) in dietary-Na results in a compensatory increase in the K concentration in saliva and subsequently in the rumen fluid and a reduction in Mg absorption. Feeding high levels of Na may increase Mg absorption, but the excessive Na supply also increases urinary excretion of both Na and Mg (28). In the

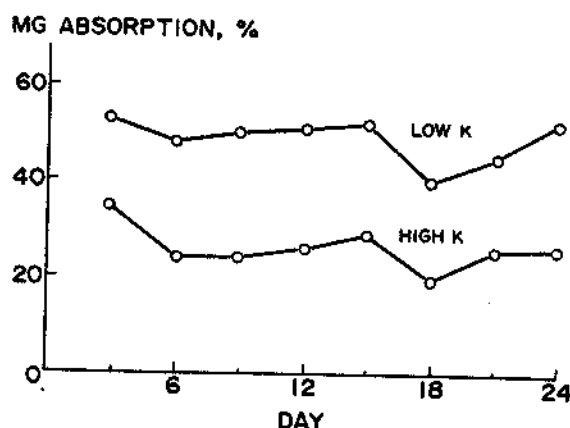


Figure 24-8. Effect of dietary K levels on apparent Mg absorption in wether lambs. Adapted from Newton et al (29).

latter case hypomagnesemia was observed despite the increased Mg absorption. Thus, the Na:K value may be more important than the absolute concentration of either Na or K (23).

The actual mechanism by which Na:K affects Mg absorption has not been identified. Martens and Kasebieter (22) suggested that, at least under in vitro conditions, Mg passes across the rumen wall by a transport system depending on the activity of the Na/K ATPase and that this activity can be reduced by high K concentrations.

Water

Martens (21) measured the Mg efflux from artificial rumen fluid in heifers 10-12 months old. He reported a near linear increase in Mg desorption from the rumen fluid at increasing Mg concentrations up to 13 mmol/l. The Mg efflux changed very little at Mg concentrations greater than that. He calculated from this study that Mg absorption was saturated when rumen fluid contained a concentration of 11 mmol/l. He noted that this value and one of 4 mmol/l previously determined for sheep, differed from results of others and attributed this to improved methodology in his studies (21).

Animals often consume large amounts of water when grazing low dry matter forage. This could present conditions (Fig. 24-6) where the free or ionized Mg concentration in the rumen fluid is much less than the

saturation value of 11 mmol/l and thus Mg absorption would be less than maximum.

Nitrogen

Grass tetany occurs most frequently when animals graze succulent forages containing high concentrations of N. Dutch, and later, New Zealand, workers reported that Mg availability decreases with increasing concentrations of N in the forage (Fig. 24-9).

The incidence of grass tetany is often accentuated by N fertilization which increases the N concentration in the forage. However, N fertilization alters the growth of forage plants and further exaggerates the levels of other dietary constituents that have been implicated in the etiology of grass tetany. These factors (Fig. 24-6) include low concentrations for dry matter, readily fermentable carbohydrates and crude fiber, and high concentrations of higher fatty acids (HFA, C12 through C18), K/(Ca + Mg) expressed on an equivalency basis, and organic acids.

The direct involvement of forage N in grass tetany may result from an interaction of high N and low fermentable carbohydrate concentrations. The rumen organisms, confronted with a rich protein and low-energy diet, metabolize the excess N into nitrogenous materials like ammonia (NH_3). The NH_3

concentrations may then rise to levels of 30-70 mmol/l in the rumen fluid. Ruminant NH_3 production correlates well ($r = .92$) with the ratio of forage N to forage water-soluble carbohydrates in cows grazing ryegrass (48). It appears that an acute rise in NH_3 concentrations has at least a temporary effect on reducing Mg absorption from the rumen (23).

Energy Deficiency

Grass tetany frequently occurs when animals graze forage that is high in digestible protein (25-30% CP), but low in digestible energy (8-12% water-soluble carbohydrates, TWSC). Grass tetany coincides with the period during which the N/TWSC values are elevated (25). If animals were able to consume a sufficient amount of the tetany-prone forage to meet their energy requirement, they would ingest up to five times their crude protein requirement much of it as non-protein N. Such animals would likely suffer from NH_3 toxicity and reduce their feed intake, leading to a lower dry matter intake and further energy deficiency. These effects reduce the formation of VFA and CO_2 and the capacity to synthesize microbial protein, leading to further elevations in rumen fluid NH_3 concentrations (23).

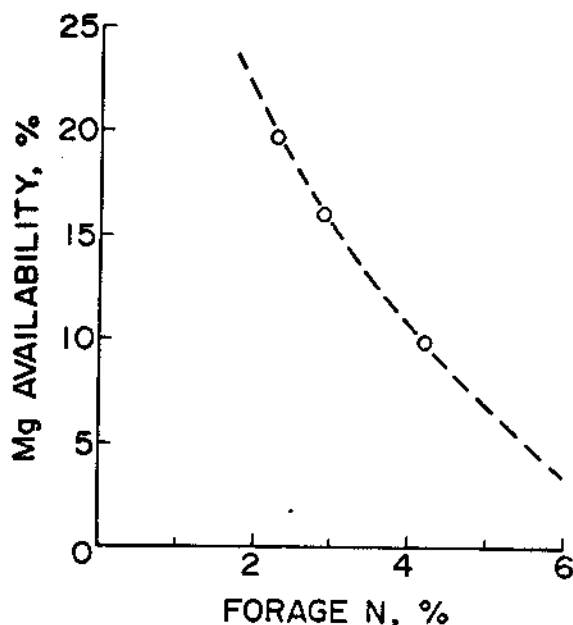


Figure 24-9. Relationship of "Mg availability" to forage N. Adapted from Metson et al (26).

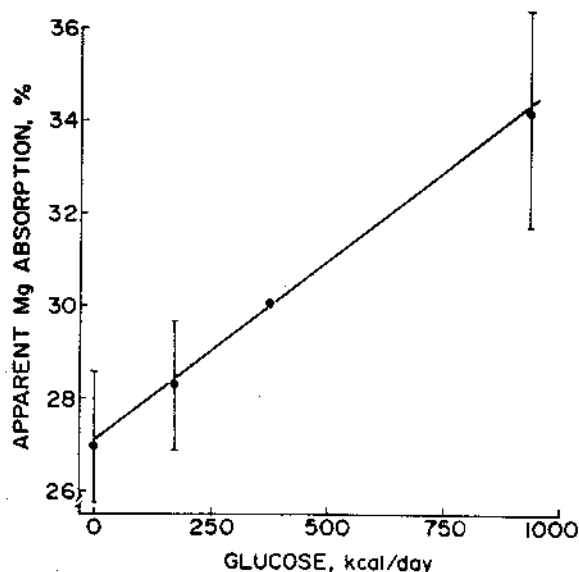


Figure 24-10. Apparent Mg absorption in sheep fed red clover-orchardgrass hay and supplemented with given levels of glucose. From Madsen et al (19).

Energy supplementation (Fig. 24-10) not only results in a higher production of VFA and CO_2 , but provides more energy for the synthesis of microbial protein. This, in turn, reduces the concentration of NH_3 and removes the inhibitory action on Mg absorption.

Higher Fatty Acids (HFA)

Forages likely to produce grass tetany in animals contain 100-200 mmol HFA/kg, largely as unsaturated palmitic, linoleic and linolenic acids. These unsaturated HFA are not absorbed in significant amounts from the rumen, but are hydrogenated and the esterified fatty acids are liberated by hydrolysis before passing with the rest of the digesta into the intestine. However, Mg and Ca may form water-insoluble soaps with the HFA, pass through the animal via the feces and thus reduce the availability of the divalent cations.

Wilson et al (53) measured blood-plasma Mg concentrations in dairy cows grazing perennial ryegrass containing 180 mmol

COOH/kg (Fig. 24-11). Cows on the control treatment suffered a significant depression in blood plasma Mg which was depressed further in cows supplemented with 50-70% additional HFA as peanut oil. A starch supplement, which served as an energy source, produced plasma Mg levels higher than those in the control group. These authors also noted that the oil supplement lowered plasma Mg concentrations more in mature than in young animals.

Organic Acids

Significant concentrations of organic acids, especially *trans*-aconitate and citrate, were measured in forages coincident to the large outbreaks of grass tetany in the 1960's. These acids were suggested as being capable of complexing Mg (10). Further animal investigations showed that drenching with KCl and either citrate or *t*-aconitate would induce tetany-like signs in animals. Drenching with only the KCl or either of the acids was ineffective (2).

It is possible that the presence of high K levels increased the absorption of the organic acid and that there may have been some complexing of Mg. However, citrate and *t*-aconitate are fermented quite rapidly in the rumen (38), and other workers have discounted the effects of these acids on Mg complexation (24). More recent research has shown that *t*-aconitate is metabolized rapidly to tricarballic acid which is fermented very slowly (38). Based on its stability constant for Mg ($K_{eq} = 115$), tricarballic acid could be a factor in hypomagnesemia (37), but in vivo verification is still needed.

Aluminum

The frequent association of high aluminum (Al) concentrations in forage and rumen samples with the incidence of hypomagnesemia has led some investigators to believe that Al could be involved with the development of grass tetany. Robinson et al (36), however, discounted the role of Al in grass tetany and reported that a large part of the Al intake was associated with soil contamination of ingested feed, and this did not affect Mg or Ca nutrition of dairy cows.

Mineral Indices

Several indices have been used to characterize the grass tetany potential of forage. The

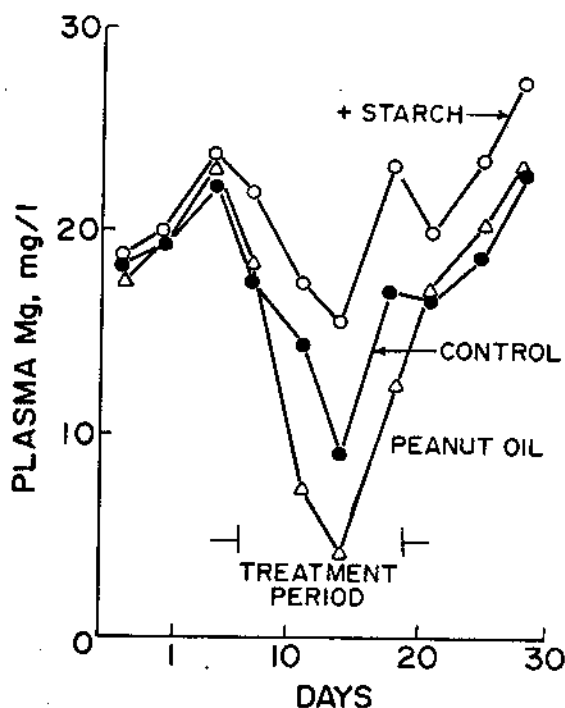


Figure 24-11. Plasma Mg concentrations in mature cows grazing a tetanigenic perennial ryegrass forage during the treatment period and not supplemented (control), or supplemented with 454 g starch or 220 ml peanut oil/d. Adapted from Wilson et al (53).

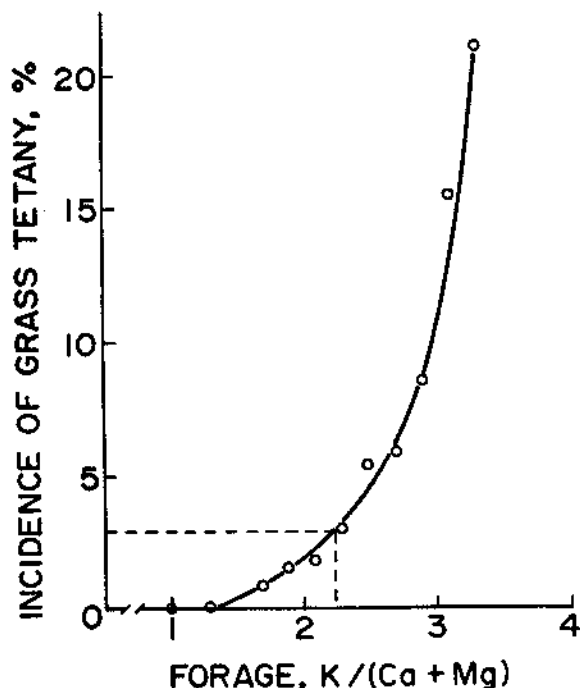


Figure 24-12. Relationship between the incidence of grass tetany and the $K/(Ca + Mg)$ in forage; calculated on an equivalence basis. Adapted from Kemp and t'Hart (17).

first and most commonly used is the $K/(Ca + Mg)$ ratio (Fig. 24-12) expressed on an equivalent basis. This ratio accounts for the antagonism of forage K and benefits of forage Mg and Ca on Mg absorption by the animal (24).

Another index, known as the Dutch Nomograph, is available in several references, including Mayland and Grunes (24). The Nomograph uses data on Mg, K and N concentrations in the forage. It is apparently useful in predicting the tetany hazard to dairy cows in areas where forage Ca levels are generally adequate (A. Kemp, personal communication). It is not as useful where forage Ca availability may be limiting. Under these conditions the $K/(Ca + Mg)$ ratio seems to be more useful.

Magnesium Activity

The contrast in Mg availability from winter dry feeds and that from spring forage is typified by the data presented by Rendig and Grunes (33). They reported that of 20-25 g Mg ingested with winter rations, 72-75% was excreted in feces. However, of the 12 g Mg ingested with spring forage, 82% was excreted

in feces. The cows on the dry feed had an apparent absorption of 6 g Mg compared with only 2 g Mg for those on the spring forage.

There is uncertainty in how Mg is bound in feedstuffs. Mg and Ca may be bound to forage particles and be relatively unavailable for adsorption by the rumen organisms or the animal. Rumen microorganisms have a much higher requirement for Mg than Ca. The washed organisms may contain 6 mg Mg/g dry matter, but only 0.1 to 0.3 mg Ca/g dry matter (5). In addition, much Mg is also adsorbed to the surface of the microorganisms. This amount increases with increasing Mg levels in the media, but decreases when rumen fluid Ca values are high. The amount of adsorbed Mg and Ca is greater at pH values ranging from 6.5 to 7.5 than in more acid conditions, which is similar to data shown in Fig. 24-13. The adsorbed Mg and Ca can be released by washing the cells in an acid solution of pH 2.5 to 3.0.

Research since 1970 (23) has shown that net Mg absorption occurs in the digestive tract proximal to the duodenum. In sheep and adult cattle most of the absorption occurs from the rumen. A small amount of Mg is absorbed from the large intestine and a small amount is secreted into the small intestine. Thus, Mg absorption occurs largely from the same section of the gastro-intestinal tract wherein the digesta is immediately exposed to several of the factors that may reduce its availability. Perhaps some of the apparent inconsistencies in treatment effects are related to pH of rumen fluid that may be made less acidic because of changes in one or more of the components like an increase in forage N/TWSC values.

The solubility and/or ultra filterability (UF) of Mg in the rumen fluid is very sensitive to pH. Smith and Horn (43) reported that under in vitro conditions the portion of UF Mg in the strained fluid was greater than 80% at an acidity less than 6.0 pH, but the portion of UF Mg was less than 20% at pH values greater than 8.0 (Fig. 24-13). The stoichiometric point is at pH 7. Thus, very small shifts in pH to less acidic conditions might well be interpreted as reducing Mg availability. Such shifts could occur by several of the factors identified in Fig. 24-6. Horn and Smith (14) found that rumen pH after feeding was as high as 7.2 when animals were fed forage

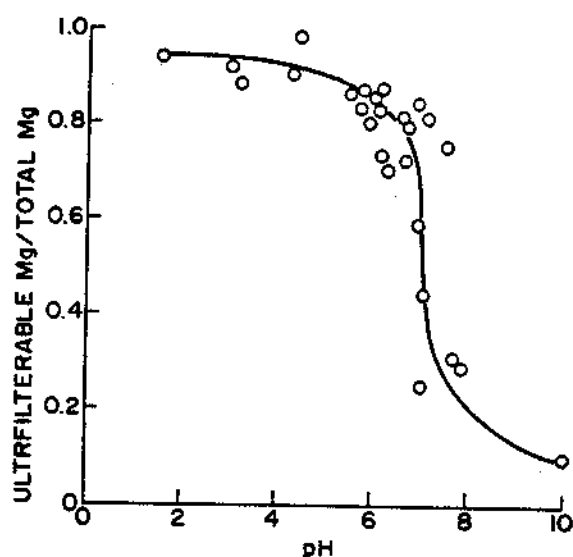


Figure 24-13. Relationship between Mg ultrafilterability and pH in rumen contents to which varying amounts of H_2SO_4 or $NaOH$ were added in vitro. From Smith and Horn (43).

and that Mg absorption proximal to the duodenum was negatively correlated with the rumen pH.

Another factor possibly affecting Mg metabolism is the ionophore which is being used more commonly to increase growth rate of ruminants including those on pasture. Serum Mg concentrations have been reduced significantly by several of these growth promotants. The ionophore may serve as a carrier of various ions across the lipid bilayer (44), but it is not known if these molecules will affect overall mg metabolism.

Another aspect of Mg activity within the lactating ruminant is its effect on milk production. Wilson (52) conducted a 3-week study with dairy cows grazing a mixed ryegrass-clover pasture. One group was drenched with 10 g Mg/d as $MgCl_2$. A second group was infused rectally with 10 g Mg/d (as $MgCl_2$) during week 1 or injected subcutaneously with 2 g Mg/d as $MgSO_4 \cdot 7H_2O$ during weeks 2 and 3. The third group served as a control. Oral, rectal and subcutaneous treatments increased ($P < .05$) plasma Mg levels about 40% above that in the control animals. Nevertheless, all animals were considered hypomagnesemic during the study. Oral supplementation, but not rectal or subcutaneous infusion, increased milk fat yield and milk

yield about 12%, leading the authors to conclude that the benefits of Mg supplementation on production were effective in the rumen fermentation process.

CLINICAL SYMPTOMS

Normal, Chronic and Acute Hypomagnesemia

Based on blood serum or plasma Mg levels, animals may be normally magnesemic (18-30 mg/l), chronically hypomagnesemic (5-18 mg/l) or acutely hypomagnesemic (<5 mg/l). Animals that are chronically hypomagnesemic may have reduced feed intake, be somewhat more nervous than usual and, if lactating, have reduced milk production. In general they do not show any visual symptoms. Acutely hypomagnesemic animals will likely exhibit visually detectable symptoms as described below. However, serum Mg levels as low as 6 mg/l in cattle have been reported without visual evidence of hypomagnesemia (12).

As with chronic hypomagnesemia, acutely hypomagnesemic animals may be normocalcemic or more often hypocalcemic. Thus, visual symptoms may be a mixture of both Mg and Ca deficiency responses. This may occur because hypomagnesemia reduces the rate of Ca mobilization (4, 39). Various stresses may alter the Mg status of the animal in a way that it progresses slowly from normal to chronic, and sometimes to acute hypomagnesemia, or that it progresses rapidly to acute hypomagnesemic tetany followed by death.

Hypomagnesemia in Cattle

Hypomagnesemia can occur in all classes of cattle, but occurs most frequently in older lactating cows. Hypomagnesemic cows exhibit signs of nervousness, reduced feed intake, reduced milk production, and may show slight muscular twitching along the face, shoulder and flank. Animals may also display some spasticity of limb muscles when walking.

In progressive clinical tetany the cows stagger and fall on their sides with their heads thrown back, salivate profusely and grind their teeth. The disorder may progress quickly to paddling with front and hind feet, fluttering of eyelids, eyeballs rolled inward, convulsions, coma and death (Fig. 24-14). There are



Figure 24-14. Lactating beef cow that has died from grass tetany. Courtesy of J.A. Stuedemann, USDA-ARS, Watkinsville, GA.

no known characteristic postmortem lesions that can be used for diagnostic purposes.

Hypomagnesemia in Sheep

In sheep there is considerable variation between animals. Generally, hypomagnesemia occurs within 2-4 weeks after sheep have been moved to improved pasture or oat forage. Ewes with twins are more susceptible to grass tetany than are ewes with single lambs (15, 49). Severely hypomagnesemic ewes are depressed, stand with their heads down, are often separated from the flock and are reluctant to move.

In progressive clinical tetany the sheep stagger, collapse on their sides with heads thrown back and some froth may be present about the mouth. They may at this time have an increased heart and respiratory rate and elevated temperature. The disorder progresses quickly to paddling with all feet, grinding teeth and generalized muscular tremors, convulsions and death. Sometimes the clinically recognizable symptoms will precede death by only a few minutes, especially when animals are chased (15). There are no known characteristic postmortem lesions, although sub-endocardial hemorrhages may occur because of the severe tetanic convulsions.

PREDISPOSING CONDITIONS FOR HYPOMAGNESEMIC TETANY

Spring Tetany or Lactation Tetany

This is the *most common* situation in which hypomagnesemia occurs. The disorder appears within 2-4 weeks after cattle or sheep have been turned out to rapidly-growing pasture. It may also occur under conditions of

autumn regrowth. Older, lactating females are at greatest risk, but the problem may occur in other classes of ruminants.

Considerable variability may exist in the blood serum Mg levels among animals in the same herd. While a few animals may have >20 mg Mg/l, the others will be evenly divided with 50% having values 11-20 and the others being <11 mg/l. Of these, two-thirds may also be hypocalcemic.

Hypomagnesemic tetany occurs in sheep grazing young perennial grass or oat forage, especially if either has been fertilized with N and/or K. Grazing such lush pastures during frosty or cold weather within 2-4 weeks after lambing seems to predispose ewes to hypomagnesemia, often with accompanying hypocalcemia (49).

Mg and many times Ca deficiencies are basic to the condition. Yet a number of factors are peculiar to the spring tetany situation including high levels of forage N, K, HFA, and organic acids and low levels of forage Na, dry matter, fiber and soluble carbohydrates. Forage Mg may sometimes be lower during this period than at later times, but in other cases Mg levels appear normal. Animals may be stressed because of lactation, cool weather, inadequate dry matter intake, estrus and high water intake.

Wheat Pasture Tetany

Animals grazing wheat pasture or other lush cereal forage may be exposed to several problems, including frothy bloat, nitrate toxicity and grass tetany. The tetany may be primarily hypocalcemia with secondary hypomagnesemia (3).

Eng (6) noted that the 10-20% death loss in cattle grazing wheat pasture in 1982-83 could be attributed to grass tetany, bloat, nitrate toxicity or enterotoxemia. The chemical composition of the forage associated with the enterotoxemia losses was 24% dry matter, 31% CP, 14% fiber, 3.4% K, 0.15% Mg and 0.68% NO₃. This profile also typifies a highly tetanogenic forage.

Winter Tetany or Seasonal Tetany

Hypomagnesemia occurring under these conditions often involves beef cattle and out-wintered dairy cattle. These animals are grazing dry, mature grass, or are being fed poor quality fodder or grass that is very low in

total Mg and other nutrients. It may also occur in stall-fed cattle on a low plane of nutrition (42). Animals may have serum Mg levels as low as 5 mg/l and varying degrees of hypocalcemia. The low serum electrolytes are often associated with adverse weather conditions.

Hypomagnesemia occurs in over-wintering cows maintained on largely straw diets and occasionally supplemented with energy and/or protein. Mg, as low as 0.6 mg/g, could limit intake and reduce performance (20). Generally, winter tetany is accompanied by few or no visual symptoms. Stress of any type may result in symptoms ranging from moderate incoordination to paresis or tetany.

The author observed winter tetany in a group of 550 pregnant beef cows pastured on corn stalks (ears removed) and supplemented with a minimal amount of poor quality grass hay. This occurred in early winter and animals were outside with no protection. Stress resulting from a period of cold windy days, parturition and very early lactation resulted in 23 deaths from hypomagnesemic tetany. The tetany stopped when high quality alfalfa hay was fed and Mg supplementation provided.

Underfeeding, Fasting or Starvation Tetany

Underfeeding associated with reduced grazing time, reduced dry matter intake, and reduced rumination time may lower serum Mg of ruminants. This may often occur when animals that are turned out to spring grass are unable to eat enough dry matter because of the short growth and low dry matter content.

Milk Tetany

This generally chronic hypomagnesemic disorder appears in calves raised on whole milk or milk replacer (unless fortified with Mg). The problem is also reported to occur in bucket-fed calves 3-4 months of age that have access to lush green spring pasture (15). Visual signs are like those reported for adult cattle.

Transportation Tetany or Transit Tetany

This condition of hypomagnesemia occurs in sheep and cattle that have undergone long periods of fasting. It is a common metabolic disease of feeder lambs that usually occurs within 10 d after arrival at the feedlot (18). The problem also occurs after prolonged

transport of cows and ewes in late pregnancy. This transportation stress may result in several disorders, including hypocalcemia, ketosis and hypomagnesemia. In the hypomagnesemia type (this may also include hypocalcemia) animals are restless, nervous and weak and have a staggering unsteady gait (Fig. 24-15). There may be partial paralysis of the hindquarters, muscular spasms, excitability, frothing at the mouth and champing of the jaws. The animal may be unable to stand and falls or lies down. It may quickly become laterally recumbent and paddle with all feet, then progress into tetanic convulsions and death.

Lucas (18) reported serum Mg, Ca, and P values (mg/l) of 14, 73 to 78 and 102 to 108, respectively, in a group of lambs where transportation tetany was occurring. He reported normal values for these minerals as 20, 110 and 70 mg/l, respectively. Repeated parenteral injections of large volumes of electrolyte solutions are recommended in addition to Mg repletion (1).

CHEMICAL SYMPTOMS

Blood serum Mg values in ruminants are normally 18-30 mg/l. Chronic hypomagnesemia is associated with serum levels of 5 to 18 mg/l and acute hypomagnesemia is associated with serum levels <5 mg/l. However, these threshold values are somewhat arbitrary (35). Levels of Mg in both blood serum and blood plasma are similar. Higher blood levels of Mg (140 mg/l) induce anesthesia, while even higher levels (200 mg/l) are toxic (42). Hypomagnesemic animals may also be normocalcemic or hypocalcemic. Blood samples drawn postmortem will have elevated Mg levels because of the release of Mg from tissues during the muscle spasms associated with the tetanic convulsions.

Urine Mg concentrations range from 0 to 280 mg/l and are curvilinearly related to blood serum Mg levels, but values approach zero as animals become increasingly hypomagnesemic. Urinary Mg levels of 20-100 mg/l are given as inadequate and <20 mg/l as indicative of severe deficiency and danger of tetany. Urine samples taken directly from the bladder within a few hours postmortem, in connection with blood samples from several



Figure 24-15. Transportation tetany in lambs. Earliest signs include stilted, ataxic gait (upper left). The next stage is lateral recumbency (upper right). Post-mortem signs include "tetany" of hind legs (lower left). The rumen may be bloated, but upon necropsy is found to be filled with fluid and empty of roughage or concentrate (lower right). Photos courtesy of R.E. Pierson (retired), Colorado State Univ., Ft. Collins, CO.

other animals in the herd, may provide a useful diagnosis.

DIFFERENTIAL DIAGNOSIS

Hypomagnesemia has often been confused with enterotoxemia. This latter condition typically occurs in calves, young cattle and sheep (15), although it has been diagnosed as occurring in adult cattle. It is caused by the toxins produced by *Clostridium perfringens* or *C. welchii* infections in the gut. It often occurs when animals are grazing lush pasture and symptoms may include staggering, salivation, convulsions and death. Death losses may be sporadic in cattle, but are often massive in sheep. Enterotoxemia can be confirmed by presence of the toxin in urine or in

gut tissue. Serum Mg levels should be determined on live animals in the herd to check the Mg status.

Grass tetany may also be confused with nitrate toxicity because both may occur on lush pastures, especially those heavily fertilized with N. The most useful indication of nitrate toxicity is the brownish discoloration of blood and tissues. Deaths from both problems have occurred together in some instances.

Some symptoms of grass tetany in high producing dairy cows may be confused with ketosis. About one-fourth of the cases will show nervous signs, stagger, froth at the mouth, have a high-stepping gait and occasionally have convulsions. Animals suffering from ketosis will have an acetone odor in

their breath and milk. The presence of ketone bodies in the urine will verify the ketosis (see earlier section).

Verification of the Mg status of ruminants is best done by measuring the Mg in blood serum obtained from at least several live animals in the herd. The Mg concentration in urine could also be used as a diagnostic test, because Mg levels decline rapidly to zero as the degree of hypomagnesemia increases. The urine sample should be carefully collected to avoid contamination with fecal material. Schneider et al (40) have proposed a quick test for evaluating the urinary Mg status of animals, but details of this procedure were not yet available to the author. This quick test may be an improvement on a semiquantitative indicator paper that has been used in Europe (41) to evaluate urine Mg levels.

TREATMENT OF ACUTE HYPOMAGNESEMIA

Treatment of tetany cases can be successful if given early and without excessive handling of the affected animals (1, 9). The safest general recommendation is to slowly inject, intravenously, 500 ml of a solution (50 ml for sheep) containing 25% Ca borogluconate and 5% Mg hypophosphite (or 15% Mg gluconate). The injections should be given slowly because there is danger of heart failure if given too rapidly. Serum Mg levels rise quickly, but return to preinjection levels within 3-6 h. A second dose may be required if the animal again exhibits acute signs. For optimum results the intravenous injection should be followed with a subcutaneous injection of 200 ml of a saturated solution of MgSO_4 (50%). Unfortunately, Mg drenches have not been effective in initially treating tetany because too much time is required for the Mg to be absorbed.

Rectally infused enemas containing 60 g of $\text{MgCl}_2 \cdot 6\text{H}_2\text{O}$ in 300 ml water have been useful in treating acute cases. Reynolds et al (34) reported that this procedure increased plasma Mg within 5 min and increased CSF-Mg levels within 30 min in calves that previously had CSF-Mg levels <18 mg/l. Administering the Mg as an enema may be easier for many livestock operators than giving intravenous or subcutaneous injections and also allows the use of non-sterile equipment.

After the initial treatment, animals should be separated from the predisposing conditions and given high quality dry hay and, if possible, energy supplements. Also 30 g Mg (2 oz calcined magnesite containing 85% MgO) should be given daily. The Mg may be administered in a drench if necessary, but the amount can be reduced after a week. Cows that get tetany are likely to get it again later in the season or in subsequent years.

PREVENTION

Animal Management Techniques

Supplementing Mg in the animal's diet is recommended. Commercial grade MgO , MgCl_2 , MgCO_3 and MgSO_4 are good sources. Dolomitic limestone and magnesite are only slightly available to the animal and are not recommended.

Animals can be fed high-Mg blocks or mineral salt mixtures. Licking wheels or licking belts are sometimes used to dispense MgSO_4 in molasses. Producers should check that the Mg remains in suspension. Avoid using crude protein-enriched supplements when cattle are grazing lush green grass.

Epsom salts ($\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$) added to the drinking water is helpful if the trough is the sole source of water. MgCl_2 or Mg acetate may also be used. The author is familiar with a beef cow-calf producer who grazes about 800 mother cows on crested wheatgrass. Fifteen to 30 animals died annually from grass tetany until Mg supplementation was initiated during the tetany period. Except for a few rain puddles, these cows rely on a single source of water which is pumped into a 5,000 gallon holding tank from which it is piped to water troughs. For several weeks prior to and during the grass tetany season he daily adds 100 lb of $\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$ to the water tank. This provides an average of 5 g Mg/cow/d and has reduced his losses to only a few animals during a 13-year period.

Spraying a water slurry of 10% MgO and 1.5% bentonite on forage is an effective way to increase Mg intake by animals grazing the treated forage. This procedure is not practical where forage yields are low.

Another practice that may be useful in preventing grass tetany is to use pastures that have been rested the previous year or have a lot of old growth present. This dry forage

tends to dilute the negative effects of the lush, green new growth.

Mg boluses, when placed in the rumen-reticulum, may be useful in some circumstances in reducing grass tetany losses. Stuedemann et al (45) reported that the type they tested dissolved at a rate of 0.66 to 1.33 g Mg/d in rumen-fistulated steers. The boluses were also placed in some mother cows. Grass tetany occurred in one control cow and in one bolus-treated cow. The authors concluded that the Mg release appears too slow to be completely effective in preventing grass tetany.

Grunes and Mayland (9) recommend that throughout the high risk period beef cattle receive 8-10 g Mg/d and that lactating cows receive 20-25 g Mg/d. They recommend that dairy cows receive 30 g Mg/d, calves receive 4-8 g Mg/d, and lactating ewes receive about 3 g Mg/d. Mg supplements should be started several weeks before the tetany period to get the animals accustomed to them. The producer should check consumption. Because the mobile Mg is lost quickly from the bodies of adult ruminants, it is necessary to supplement on a daily basis during the tetany period (35).

Agronomic Management Techniques

Agronomic steps that will reduce the incidence of tetany include: (a) grazing forage having a higher level of available Mg, (b) applying moderate amounts of N and K fertilizer and then in split applications, (c) applying dolomitic limestone rather than calcitic limestone, if liming is required and (d) using mixed grass-legume pastures where possible.

Selection and breeding work is underway to develop tall fescue and crested wheatgrass cultivars that impose a lower risk of tetany (author's laboratory). Some selection work has already been conducted on Italian ryegrass. Moseley and Griffiths (27) fed sheep two experimental populations of Italian ryegrass. The one has 1.7 mg Mg/g and 4.6 mg Ca/g, while the other had values of 2.4 and 5.6, respectively. Concentrations of other minerals and IVDMD were not different between the two selections. Mg intake, apparent availability and retention were significantly greater with the high-Mg selection. Rumen fluid from animals fed the high-Mg

selection had a higher proportion of soluble Mg and a higher Na:K ratio in the supernatant liquor.

Reid et al (32) evaluated the effect of Mg supplementation of sheep when fed timothy grass. The grass was grown on a coarse textured acid soil that was either not fertilized or received 390 kg Mg/ha. Magnesium fertilization increased forage Mg concentration from 0.8 mg/g in the control to 1.5 mg/g. Supplemental feeding of Mg to sheep increased the intake of unfertilized as well as fertilized timothy by 13 and 15%, respectively. In this study, IVDMD of the Mg fertilized grass was less than that of the unfertilized grass. The Mg fertilization and the Mg supplementation of sheep increased Mg availability and serum Mg concentrations.

SUMMARY

Grass tetany is a major problem of cattle and sheep in temperate climates. It results from an inadequate supply of available Mg. This occurs when either actual intake of Mg is limited or, more often, because factors in the diet reduce the Mg availability. Grass tetany may also occur because of a need for increased amounts of Mg during parturition and early lactation.

Although grass tetany occurs in all classes of ruminants, older lactating animals are at greatest risk because of a reduced ability to mobilize body reserves of Mg. Chronic hypomagnesemia results in reduced feed intake and milk production. It may progress quickly into acute hypomagnesemia which terminates in convulsions, coma and death. Acutely stricken animals should receive Mg via enema or, preferably, by intravenous or subcutaneous injection. Chronically hypomagnesemic animals should receive daily supplemental Mg. Hypomagnesemic animals are frequently hypocalcemic.

URINARY CALCULI

by R. J. Emerick

Urinary blockage by calculi is an important cause of deaths in cattle and sheep. Occasional reports of the disease in other ruminants indicate that they are similarly

Literature Cited on Grass Tetany

1. Blood, D.C., J.A. Henderson and O.M. Radostits. 1979. *Veterinary Medicine*. Lea and Febiger, Philadelphia.
2. Bohman, V.R., A.L. Lesperance, G.D. Harding and D.L. Grunes. 1969. *J. Animal Sci.* 29:99.
3. Bohman, V.R., F.P. Horn, E.T. Littledike, J.G. Hurst and D. Griffin. 1983. *J. Animal Sci.* 47:1364.
4. Contreras, P.A., R. Manston and B.F. Sansom. 1982. *Res. Vet. Sci.* 33:10.
5. Durand, M. and R. Kawashima. 1980. In: *Digestive Physiology and Metabolism in Ruminants*. Y. Ruckebusch and P. Thivend, eds. AVI Publ. Co., Westport, CT.
6. Eng, K. 1983. *Feedstuffs* 25:10.
7. Fontenot, J.P., G.E. Bunce, K.E. Webb, Jr. and V.G. Allen (eds.). 1983. *Role of Magnesium in Animal Agriculture*. John Lee Pratt Animal Nut. Pro., Virginia Polytechnic Institute, Blacksburg, VA.
8. Grace, N.D. 1983. *The Mineral Requirements of Grazing Ruminants*. N. Z. Soc. Animal Prod. Occasional Publ. No. 9.
9. Grunes, D.L. and H.F. Mayland. 1984. USDA Leaflet No. 561.
10. Grunes, D.L., P.R. Stout and J.R. Brownell. 1970. *Adv. Agron.* 22:331.
11. Grunes, D.L., J.F. Thompson, J. Kubota and V.A. Lazar. 1968. *Ninth Intl. Congr. Soil Sci. Trans.* II:597.
12. Hidioglu, J., B.K. Thompson, S.K. Ho and J.G. Proulx. 1981. *Can. J. Comp. Med.* 45:124.
13. Hjerpe, C.A. 1967. *Southwest. Vet.* 21:33.
14. Horn, J.P. and R.H. Smith. 1978. *Brit. J. Nutr.* 40:473.
15. Hungerford, T.G. 1975. *Diseases of Livestock*, 8th Ed. McGraw-Hill Book Company, Sydney.
16. Jolley, L.C. and D.D. Leaver. 1974. *Aust. Vet. J.* 50:98.
17. Kemp, A. and M.L. t'Hart. 1957. *Neth. J. Agr. Sci.* 5:4.
18. Lucas, M.J. 1983. *Mod. Vet. Prac.* 64:213.
19. Madsen, F.C., D.E. Lentz, J.K. Miller, D. Lowrey-Harnden and S.L. Hansard. 1976. *J. Animal Sci.* 42:1316.
20. Mathison, G.W., R.T. Hardin and B.E. Beck. 1981. *Can. J. Animal Sci.* 61:375.
21. Martens, H. 1983. *Brit. J. of Nutr.* 49:153.
22. Martens, H. and H. Kasebieter. 1983. *Zbl. Vet. Med. A*, 30:1.
23. Martens, H. and Y. Rayssiguier. 1980. In: *Digestive Physiology and Metabolism in Ruminants*. Y. Ruckebusch and P. Thivend, eds. AVI Publ. Co., Westport, CT.
24. Mayland, H.F. and D.L. Grunes. 1979. In: *Am. Soc. Agron. Publ. 35*, p. 123. V.V. Rendig and D.L. Grunes, eds. Madison, WI.
25. Mayland, H.F., D.L. Grunes and D.M. Stuart. 1974. *Agron. J.* 66:441.
26. Metson, A.J., W.H.M. Saunders, T.W. Collie and V.W. Graham. 1966. *N. Z. J. Agr. Res.* 9:410.
27. Moseley, G. and D.W. Griffiths. 1984. *Grass and Forage Sci.* 39:195.
28. Moseley, G. and D.J.H. Jones. 1974. *Proc. Nutr. Soc.* 33:87A.
29. Newton, G.L., J.P. Fontenot, R.E. Tucker and C.E. Polan. 1972. *J. Animal Sci.* 35:440.
30. O'Kelley, R.E. and J.P. Fontenot. 1969. *J. Animal Sci.* 29:959.
31. O'Kelley, R.E. and J.P. Fontenot. 1973. *J. Animal Sci.* 36:994.
32. Reid, R.L., B.S. Baker and L.C. Vona. 1984. *J. Animal Sci.* 59:1403.
33. Rendig, V.V. and D.L. Grunes (eds.). 1979. *Grass Tetany*. Am. Soc. Agron. Publ. 35. Madison, WI.
34. Reynolds, C.K., M.C. Bell and M.H. Sims. 1984. *J. Nutr.* 114:1334.
35. Ritter, R.J., J.A. Boiling and N. Gay. 1984. *J. Animal Sci.* 59:197.
36. Robinson, D.L., O.J. Hemkes and A. Kemp. 1984. *Netherlands J. Agr. Sci.* 32:73.
37. Russell, J.B. 1985. *Appl. Environ. Microbiol.* 49:120.
38. Russell, J.B. and P.J. Van Soest. 1984. *Appl. Environ. Microbiol.* 47:155.
39. Sansom, B.F., R. Manston and M.J. Vagg. 1983. *Vet. Record* 112:447.
40. Schneider, E., P. Tschudi and W. Leuenberger. 1985. *Schweizer Archiv fur Tierheilkunde*. 127:9.
41. Simesen, M.G. 1968. *Medlemsblad Danske Dyrlaegeforen*. 51:848.
42. Simesen, M.G. 1970. In: *Clinical Biochemistry of Domestic Animals*, 2nd Ed., Vol. 1:353. J.J. Kaneko and C.E. Cornelius, eds. Academic Press, New York.
43. Smith, R.H. and J.P. Horn. 1976. In: *Nuclear Techniques in Animal Production and Health*. IAEA. Vienna.
44. Spears, J.W. and R.W. Harvey. 1984. *J. Animal Sci.* 58:460.
45. Stuedemann, J.A., S.R. Wilkinson and R.S. Lowrey. 1984. *Amer. J. Vet. Res.* 45:698.
46. Terashima, Y., et al. 1984. *Nutr. Rep. Int.* 29:869-875.

47. Turner, M.A. and V.E. Neall. 1978. N. Z. J. Agr. Res. 21:583.
48. Verite, R., B. Remond and M. Journet. 1984. Can. J. Animal Sci. 64 (Suppl):328.
49. West, D.M. and A.N. Bruere. 1981. N. Z. Vet. J. 29:85.
50. Whitaker, D.A. 1983. Outlook on Agr. 12:77.
51. Wilkinson, S.R. and J.A. Stuedemann. 1979. In: Am. Soc. Agron. Publ. 35, p. 93. V.V. Rendig and D.L. Grunes, eds. Madison, WI.
52. Wilson, G.F. 1980. Animal Prod. 31:153.
53. Wilson, G.F., et al. 1969. N. Z. J. Agr. Res. 12:467.

Literature Cited on Urinary Calculi

1. Emerick, R.J. and L.B. Embry. 1964. J. Animal Sci. 23:1079.
2. Nottle, M.C. 1982. Aust. Vet. J. 58:256.
3. Nottle, M.C. 1976. Res. Vet. Sci. 21:309.
4. Huntington, G.B. and R.J. Emerick. 1984. Amer. J. Vet. Res. 45:180.
5. Walter-Toews, D. and D.H. Meadows. 1980. Can. Vet. J. 21:61.
6. Finlayson, B., C.W. Vermeulen and E.J. Stewart. 1961. J. Urol. 86:355.
7. Packett, L.V. and S.D. Coburn. 1965. Amer. J. Vet. Res. 110:112.
8. Hoar, D.W., R.J. Emerick and L.B. Embry. 1969. J. Animal Sci. 29:647.
9. Lindley, C.E., et al. 1953. J. Animal Sci. 12:704.
10. Emerick, R.J., L.B. Embry and O.E. Olson. 1959. J. Animal Sci. 18:1025.
11. Emerick, R.J. and L.B. Embry. 1963. J. Animal Sci. 22:510.
12. Bushman, D.H., R.J. Emerick and L.B. Embry. 1965. J. Animal Sci. 24:671.
13. Bushman, D.H., R.J. Emerick and L.B. Embry. 1965. J. Nutr. 87:499.
14. Bushman, D.H., L.B. Embry and R.J. Emerick. 1967. J. Animal Sci. 26:1199.
15. Bushman, D.H., R.J. Emerick and L.B. Embry. 1968. J. Animal Sci. 27:490.
16. Hoar, D.W., R.J. Emerick and L.B. Embry. 1970. J. Animal Sci. 31:118.
17. Packett, L.V. and J.P. Hauschild. 1964. J. Nutr. 84:185.
18. Godwin, I.R. and V.J. Williams. 1982. Aust. J. Agr. Res. 33:843.
19. N.R.C. 1964. Joint United States-Canadian tables of feed composition. Nat. Acad. Sci., Washington, DC.
20. Crookshank, H.R., et al. 1960. J. Animal Sci. 19:595.
21. Elam, C.J., W.E. Ham and B.H. Schneider. 1957. Proc. Soc. Expt. Biol. Med. 95:769.
22. Connell, R., F. Whiting and S.A. Forman. 1959. Can. J. Comp. Med. 23:41.
23. Keeler, R.F. 1963. Annals N. Y. Acad. Sci. 104:592.
24. Herman, J.R. and A.S. Goldberg. 1960. J. Amer. Med. Assoc. 174:128.
25. Emerick, R.J., E.E. Kugel and V. Wallace. 1963. Amer. J. Vet. Res. 24:610.
26. Emerick, R.J. 1984. J. Nutr. 114:733.
27. Bailey, C.B. 1981. Can. J. Animal Sci. 61:219.
28. Fransen, S.C. 1982. Ph.D. Thesis. South Dakota State Univ., Brookings, SD.
29. Bailey, C.B. 1967. Amer. J. Vet. Res. 28:1743.
30. Healy, W.B. 1973. Nutritional aspects of soil ingestion by grazing animals. In: Chemistry and Biochemistry of Herbage, Vol. 1. Academic Press, London.
31. Schneider, B.H., E.D. Tayson and W.E. Ham. 1952. Wash. Agr. Exp. Sta. Cir. 203.
32. Huntington, G.B., R.J. Emerick and L.B. Embry. 1977. J. Animal Sci. 45:119.
33. Jones, L.H.P. and K.A. Handreck. 1965. J. Agr. Sci. 65:129.
34. Scheel, L.D., E. Fleischer and F.W. Klemperer. 1953. Ind. Hyg. and Occupational Med. 8:564.
35. Bailey, C.B. 1976. Can. J. Animal Sci. 56:213.
36. Nottle, M.C. 1966. Aust. J. Agr. Res. 17:175.
37. Nottle, M.C. 1966. Ausf. J. Agr. Res. 17:183.
38. Iler, R.K. 1955. The Colloid Chemistry of Silica and Silicates. Cornell Univ. Press. Ithaca, NY.
39. Merrill, R.C. and R.W. Spencer. 1950. J. Phys. and Colloid Chem. 54:806.
40. Bailey, C.B. 1972. Can. J. Biochem. 50:305.
41. Whiting, F., R. Connell and S.A. Forman. 1958. Can. J. Comp. Med. 22:332.
42. Bailey, C.B. 1967. Science (N.Y.) 155:696.
43. Bailey, C.B. 1977. Can. J. Animal Sci. 57:239.